Diagnostic Ophthalmology Ophtalmologie diagnostique

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History and clinical signs

13-year-old, neutered male, Siberian husky was examined by the ophthalmology service at the Western College of Veterinary Medicine. This dog was being treated with radiation therapy by the oncology service (WCVM) for a soft tissue sarcoma. The presenting complaint was an anisocoria that had been present for a few days. Physical examination confirmed right miosis, enophthalmos, and ptosis (Figure 1). The menace responses, and the palpebral, pupillary light, and oculocephalic reflexes were present in both eyes. Schirmer tear test (Schirmer Tear Test Strips; Alcon Canada, Mississauga, Ontario) values were within normal reference ranges in both eyes. The intraocular pressures, estimated with an applanation tonometer (Tonopen XL; Biorad Ophthalmic Division, Santa Clara, California, USA), were 16 and 21 mmHg in the right and left eyes, respectively. The pupils were dilated with tropicamide (Mydriacyl; Alcon Canada); the right pupil dilated only marginally to about 4 mm, while the left pupil dilated completely. Biomicroscopic examination (Osram 64222; Carl Zeiss Canada, Don Mills, Ontario) confirmed bilateral incipient anterior cortical cataracts. Examination of the posterior segments of both globes with an indirect ophthalmoscope (Heine Omega 200; Heine Instruments Canada, Kitchener, Ontario) failed to identify posterior segment abnormalities.

What are your clinical diagnoses, differential diagnoses, diagnostic plan, and prognosis?

Our tentative clinical diagnosis, based on the miosis, enophthalmos, ptosis, and a lack of detectable intraocular disease, was Horner's syndrome in the right eye. The minimal dilatation after topical topicamide also supported the diagnosis of right eye Horner's syndrome. However, mild subclinical uveitis could not be completely excluded. The topical ocular 5% cocaine response test is required to confirm Horner's syndrome. If confirmed, the syndrome should be localized as pre- or postganglionic. We advised a reexamination to evaluate the response of both pupils to scotopic and photopic conditions and to confirm that the right pupil was the abnormal pupil. Forty-eight hours later, we noted that the anisocoria lessened in bright photopic conditions and worsened in scotopic conditions and that the most actively moving pupil was on the left. The lesser mobile

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Figure 1. A 13-year-old Siberian husky with right enophthalmos, ptosis, miosis, and nictatans prolapse.



Figure 2. The Siberian husky in Figure 1, approximately 3 months later. Note the bilateral miosis and prolapsed nictatans.

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right pupil was most likely the abnormal one. We completed a topical cocaine response test and the left pupil dilated, while the right miosis, 3rd eyelid prolapse and enophthalmos, and ptosis remained; these findings confirmed the diagnosis of right eye Horner's syndrome (1). We advised and completed a dilute adrenergic pupillary response test 48 h later, using 0.1% phenylephrine (Bausch & Lomb Canada, Markham, Ontario) in both eyes (1). When no response was noted in either pupil, we completed a concentrated 10% phenylephrine response test in both eyes, which was positive (dilation of both pupils) in 20 min, thus confirming a preganglionic Horner's syndrome. We recommended and completed cervical and chest radiographs to exclude metastatic or primary mediastinal or cervical metastatic neoplasia and non-neoplastic tumors. None were identified. A few days later the left eye also developed signs of Horner's syndrome (Figure 2). We recommended and completed computerized tomography of the skull, neck, and chest; however, abnormalities along the vagosympathetic trunk and vertebral column were not identified. The signs of bilateral Horner's syndrome continued until the dog's death, approximately 8 mo later, due to an unrelated disorder; a postmortem examination was not completed.

Our final diagnosis was bilateral preganglionic Horner's syndrome. Horner's syndrome develops most commonly in dogs, secondary to postganglionic lesions of the sympathetic nervous system due to inflammatory disorders around and affecting the middle ear (2,3). Preganglionic lesions inducing Horner's syndrome are less common and due often to a mediastinal tumor, but occasionally to cervical vagosympathetic trunk injuries related to inadvertent damage during a jugular venous puncture, trauma, or surgical procedures. Central lesions inducing Horner's syndrome are rare and usually associated with neoplastic or inflammatory disorders of the central nervous system, when multiple neurologic defects will be present. Horner's syndrome is usually diagnosed in veterinary practice based on the clinical signs of miosis, enophthalmos, 3rd eyelid prolapse, and ptosis. However, these clinical signs are nonspecific and common with uveitis, orbital disorders, ulcerative and nonulcerative keratitis, conjunctival foreign bodies, etc. Therefore, clinical examinations must rule out these common disorders, and Horner's syndrome should be confirmed and then localized as postganglionic, preganglionic, or central. Complete biomicroscopic and indirect ophthalmoscopic examination, tonometry, Schirmer tear tests, and fluorescein staining will confirm or negate differential diagnoses like uveitis, keratitis,

conjunctival foreign bodies, and orbital disorders. However, to confirm the diagnosis of Horner's syndrome, no dilatation after the application of topical cocaine is the gold standard. Subtle dilatation of a miotic pupil to mid-size after the application of mydriatics supports the diagnosis.

Once Horner's syndrome has been confirmed by a lack of dilatation of the affected pupil by topical cocaine, the lesion should be localized. Localization of a Horner's syndrome is initiated by topical application of dilute topical adrenergics (0.1% epinephrine or phenylepinephrine) to both eyes to determine if a hyper-response is detectable in the miotic pupil within 20 min (1). No response is expected in the normal pupil, and it is important to ensure that the dilute adrenergic agent does not stimulate the normal iris dilator. If the Horner's pupil dilates within 20 min, while the nonaffected fails to dilate, the lesion is postganglionic, somewhere between the sympathetic ganglion (near the base of ear) and the iris on the ipsilateral side. If the affected and the nonaffected pupils fail to dilate within 20 min, concentrated adrenergic (10% phenylephrine) should be applied to the corneas, and if dilatation of both pupils occurs within 20-40 min, the lesion is most likely preganglionic (somewhere between the base of the ear down the vagosympathetic trunk to the cranial mediastum). Horner's syndrome localized in a preganglionic location should have cervical and thoracic radiographs completed to rule out mediastinal and cervical tumors, which are the most common etiology in dogs.

The Horner's syndrome in this Siberian husky was localized as bilateral preganglionic. However, no lesions could be identified on advanced imaging of the skull, neck, or chest. Iatrogenic vagosympathetic trauma during jugular sampling affecting both sympathetic trunks was considered most likely in this dog. Approximately 8 mo later, this dog was euthanized, due to the onset of lethargy and anemia. The signs of bilateral Horner's syndrome were still present. The cause of the anemia and lethargy was not determined, as a postmortem examination was not completed.

References

- 1. Zimmerman CF. Drugs for the diagnosis of pupillary disorders. In: Zimmerman TJ, Koonere KS, Fechtner RD, Sharir M. Textbook of Ocular Pharmacology. Hagerstown, Maryland: Lippincott-Raven Publ, 1997:827–835.
- Kern TJ, Aromando MC, Erb HN. Horner's syndrome in dogs and cats: 100 cases (1975–1985). J Am Vet Med Assoc 1989;195:369–373.
- van den Broek AHM. Horner's syndrome in cats and dogs: A review. J Small Anim Pract 1987;28:929–940.

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